Table 2: Summary of Studies concerning Biological effects of Low Frequency Modulation of RF Radiation.

Effects	Species	RF (MHz)	Mod^n (Hz)	Intensity (mW/cm^2)	Time (min)	SAR (W/kg)	Reference
Altered calcium-ion efflux in brain tissue in vitro:							
Frequency specificity	Chicken	147	6-20	1-2	20	0.002*	Bawin et al.(1975)
influence of pH and lanthanum	Chicken	450	16	0.75	20	0.0035	Bawin et al.(1978)
frequency and intensity specificity	Chicken	147	16	0.83	20	0.0014	Blackman et al.(1979)
intensity specificity and sample spacing	Chicken	147	9,16	0.083	20	0.0014	Blackman et al.(1980a)
intensity specificity and sample spacing	Chicken	147	16	0.083	20	0.0014	Joines et al (1981)
intensity specificity	Chicken	450	16	0.1-1	20	0.005-0.005	Sheppard et al.(1979)
two intensity ranges	Chicken	50	16	1.5 3.6	20 20	0.0013 0.0035	Blackman et al.(1980b)
theoretical analysis of RF dependence	Chicken	50 147 450	16	-	20	~0.001	Joines and Blackman(1980) Athey (1981); Joines and Blackman (1981).
test of predictions of theoretical analyses	Chicken	147	16	0.37 0.49	20	0.0006 0.0008	Blackman et al.(1981)
no effect for pulse modulation	Rat	1000	16,32	0.5-15	20	0.15-4.35	Shelton and Merritt (1981)
no effect for pulse modulation	Rat	1000 2450	16 8,16,32	1,10 1	20	0.29-2.9 0.3	Merrit et al. (1982)
change in calcium efflux kinetics in synaptosomes	Rat	450	16	0.5	10	-	Lin-Liu and Adey (1982)
frequency and intensity specificity in cultured neuroblastoma cells	Human being	915	16	-	30	0.05	Dutta et al.(1984)
Altered calcium ion efflux in brain tissue in vivo:							
no effect for pulse mod.	Rat	2060	8,16,32	0.5-10	20	0.12-2.4	Merritt et al.(1982)
change in efflux kinetics	Cat	450	16	3	60	0.29	Adey et al.(1982)
Changes found in pancreatic slices	Rat	147	16	2	60-150	<0.075	Albert et al.(1980)
Changes in Hearts	Frog	240	0.5,16		30	0.00015-	Schwartz et al (1990)

Electromagnetic fields, through their effect on calcium ions, play a vital role in the immune system, Walleczek (1992). Walleczek (1992) quotes research relating to the role of calcium, sodium and potassium ions, including research showing that EMF could alter the activity of the membrane incorporated Ca^{2+}-ATPase responsible for pumping Ca^{2+} out of the cell (calcium ion efflux).

Also that data from two laboratories demonstrate that ELF fields alter the activity of another membrane ion pumps, Na^{+}/K^{+}-ATPase with current densities as low as 50$\mu A/cm^2$ and estimated, by the authors, to also have an effect at 1$\mu A/cm^2$. At 50$\mu A/cm^2$, J = 0.5 A/m^2; E=2.5 V/m, assuming σ=0.2 S/m. Hence from Eq.(3) S= 1.7 $\mu W/cm^2$ and SAR = 0.00063 W/kg from Eq.8 . If the extrapolation to 1$\mu A/cm^2$ is confirmed then the EMR effects will be occurring at $1/2500^{th}$ of the S and SAR levels estimated here.

This demonstrates the extremely low induced currents, SARs and energy densities which are associated with EMR induced changes in ion pumping and calcium, sodium and potassium efflux at the cellular level.

Walleczek and Budinger (1992) report that:

> **"To date, at least 10 different laboratories, including our own, have reported ELF magnetic influences on lymphoid cells, and stimulatory as well as inhibitory effects on parameters related to calcium metabolism or RNA- and DNA-synthesis have been observed."**

They also state that:

> **"A plausible magnetic interaction mechanism based on radical pair recombination reactions which are linked to cellular signal transduction and application processes has been proposed (Grundler et al. (1992)). Magnetic field intensities similar to the intensities used in most experiments (e.g. 1-30 mT) are known from magnetochemistry to be able to influence *non-thermally* the kinetics and product yields from radical pair reactions *in vitro*, Steiner at al. (1989). The underlying reaction scheme is well known and is described by the radical pair mechanism.**
>
> **For this mechanism to be applicable to the data reported here, a pathway by which magnetically-sensitive radical-dependent processes could influence mitogen-induced lymphocyte Ca^{2+} signaling must be postulated. There is new evidence that such pathways exist.**
>
> **For example, Con A-induced Ca^{2+} uptake in rat thymic lymphocytes has been shown to depend on the generation of reactive oxygen radical species. There is also evidence from inhibition studies that cytochrome P-450 activity may be involved in Ca^{2+} uptake regulation in rat thymic lymphocytes, Alvarez et al. (1992), and it is known that P-450 function proceeds via radical pair recombination steps, Hollenberg (1992). Thus it is plausible to investigate if externally applied magnetic fields may interfere with radical pair reactions and as a consequence, may alter lymphocyte Ca^{2+} regulation."**

Calcium ion influx has been shown to play a role in the transcript levels of proto-oncogenes c-myc and c-fos which alters in the presence of electromagnetic fields, Karabakhtsian et al (1994).

Lindstrom et al. (1995) replicate and extend the research of Walleczek (1992), using the T-cell line (lymphocytes) for human leukaemia cells, and show that oscillating low-level magnetic fields produce the same calcium ion reaction as does an antibody. They show that weak magnetic fields initiate calcium ion oscillations with a threshold flux density of 40 μT, a plateau at 150 μT and a frequency range from 5 to 100 Hz, with a fairly broad peak at 50 Hz.

Galvanovskis et al. (1996) report significant reductions (30 %) in the calcium ion oscillation amplitude in human leukaemia T-cells when exposed to 50 Hz magnetic fields.

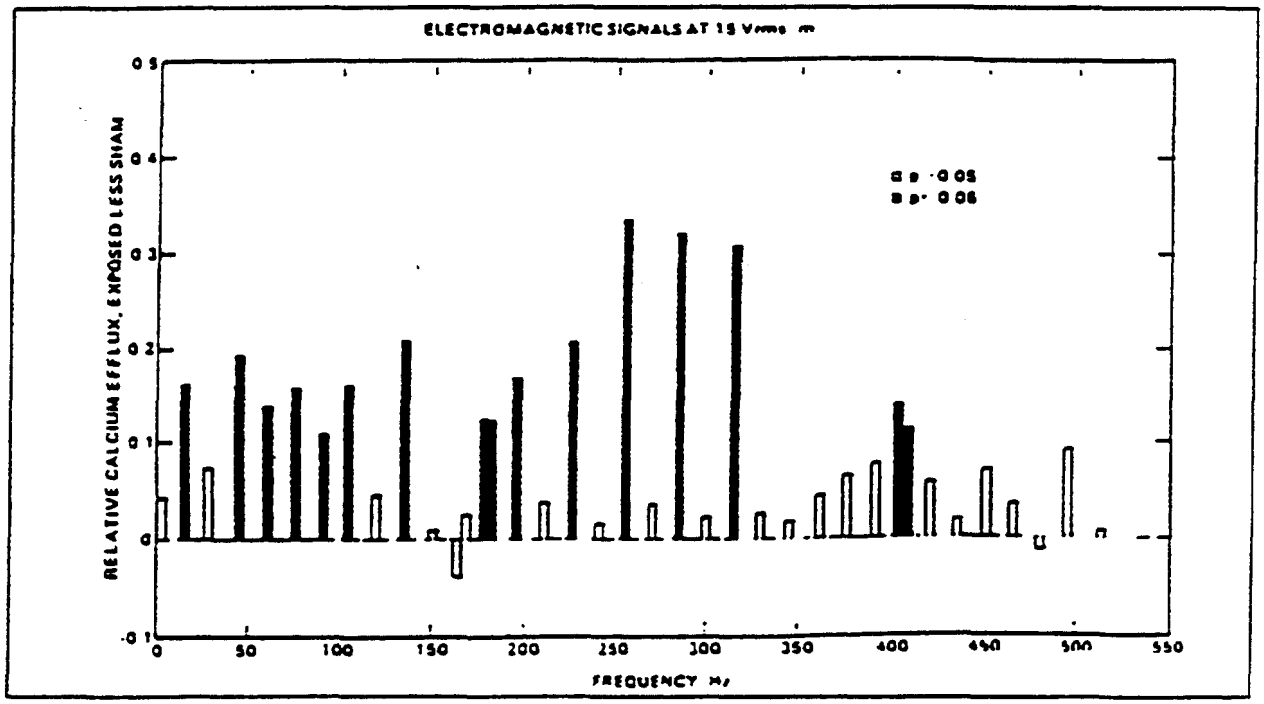

Figure 18: The effect of 15 V/m electromagnetic fields on the efflux of calcium ions from chicken brain tissue as a function of modulation frequency. The relative efflux is the difference between exposed and unexposed samples. The data from 1 to 120 Hz are taken from Blackman et al. (1985). Blackman et al. (1988).

The key role of modulation frequency in the alteration of calcium ions was recognised early. A leading researcher in this area, Dr Carl Blackman of the U.S.E.P.A. has shown that research has identified modulation frequencies which significantly alter calcium ion efflux out to 510 Hz, Figure 18.

This takes us away from the long term concentration on the 16 Hz calcium ion oscillation which first attracted attention. We can only speculate on what the results would have been in the above quoted experiments if the modulation of ELF frequency had been extended out to 500 Hz.

Their research further shows the involvement of polypeptide molecules, specifically poly-L-lysine, which the authors postulate may explain the intracellular calcium ion EMR effects on cell membrane surfaces, through the polylysine causing strong deformations on the cell surface which could trigger the release of stored calcium cations from intracellular pools, thus starting the oscillations. The authors conclude:

> **"These results allow us to suggest that 50 Hz; 100 μT magnetic fields might influence some step in the chain of biochemical events leading to the sustained calcium ion oscillation."**

They further note:

> **"That more than 20 enzymes are thought to incorporate radical chemistry in the conversion of substrates to products. It is possible that some enzymes or intermediates containing radicals are involved in the complex system responsible for intracellular calcium ion regulation. It has been shown that such biochemical reactions may be sensitive to the magnetic field."**

6.3 Intensity and Frequency Windows:

Calcium ion efflux is shown to be enhanced in intensity and modulation frequency windows, suggesting a pseudo-quantum effect. The early work of Dr Susan Bawin and Professor Ross Adey, Bawin and Adey (1976) and Adey (1980) for example, identified differences with exposure intensity and modulation frequency suggesting intensity and frequency "windows" in relation to calcium ion efflux from chick brains. Much of this work was replicated by Dr Carl Blackman at the U.S.E.P.A., e.g. Blackman et al. (1989). In Blackman's paper he comments that they could not replicate the Bawin and Adey results until his team had carefully examined the power-density dependence of the field and discovering that only certain power densities and certain modulation frequencies were capable of eliciting the response. Blackman et al. (1989) found that at 16 Hz modulation of a 50 MHz carrier with a highest SAR of 0.005 W/kg. They report that statistically significant effects were found at power densities of 1.44-1.67, 1.75, 3.85, 5.57, 6.82, 7.65, 7.77, and 8.82 mW/cm^2, but not at 0.37, 0.72, 0.75, 2.17, 2.30, 4.32, 4.50, 5.85, 7.08, 8.19, 8.66, 10.6, and 14.7 mW/cm^2 .

Blackman et al. (1989) propose a fractal process with a non-integer dimension of 1.4 to explain a series of highly peaked responses which correspond to cell membrane level amplicational processes. Using the probability of the statistical significance (p) which is $p<0.001$ for the strong peaks, they note that there is no decrease at the lower power densities making it impossible to extrapolate to a lowest threshold. Dr Adey has shown nonlinear dynamical responses at the cellular level for some time, but Dr Blackman and his group claim to be the first to apply fractal geometry to the problem and in doing so, open the possibility for functional alterations to the CNS due to very weak stimuli.

The lowest reported SARs with statistically significant increases in calcium ion efflux at 0.00015 and 0.0003 W/kg from Schwartz et al. (1990), using 16 Hz modulation and a 240 MHz carrier.

Blackman et al. (1991) define a temperature window for the calcium ion efflux from avian brain tissue. Effects are seen for 36 and 37°C but not for 35 and 38°C. The effects are evident within the normal core temperature range but not outside it.

These results, power density and temperature windows, explain why no effects were seen in early experiments which used high power densities and raised the temperature of the sample. It is not a simple matter of higher exposure gives greater effects, i.e. there is not a simple dose-response relationship. The effects are highly quantized by particular sets of conditions which trigger cell membrane reactions which involve enzyme amplifiers in the signal transduction process which are thought to be poised at a phase or cooperative transition.

6.4 Calcium-ion Signaling Summary:

ELF and RF/MW modulated at ELF frequencies, change the oscillation frequency and amplitude and they change the influx and efflux of calcium ions in and around the cell membrane.

The changing oscillation frequency and amplitude is related to the immune response of the cell and shows that the oscillating applied field produces an antibody-like reaction as though the cell has been attacked.

The influx and efflux changes relate to the signal transduction pathway in which calcium ions participate. This is one of the biochemical pathways which regulate cell behaviour. This is altered by the applied oscillating electromagnetic field. Since signal transduction controls the cell division, cell differentiation and cell proliferation, this EMR induced alteration to signal transduction has the strong potential to participate in tumour formation or promotion. Alteration of T-lymphocytes and other immune system factors suggests that EMR exposure causes immuno-suppression, partly through induced calcium ion efflux.

The following section on DNA damage and chromosome aberrations is consistent with this. While research shows that DNA is damaged and chromosome aberrations are found in EMR exposed cells, animals and people, the evidence does not point to direct DNA breakage but to the involvement of free radicals or some other cellular level mechanism such as altered signal transduction pathways.

6.5 Calcium ion Conclusion:

Courtesy of Professor Ross Adey, Adey (1993):

> **"Life on earth has evolved in a sea of natural electromagnetic (EM) fields. Over the past century, this natural environment has sharply changed with the introduction of a vast and growing spectrum of man-made EM fields. from models based on equilibrium thermodynamics and thermal effects, these fields were initially considered too weak to interact with biomolecular systems, thus incapable of influencing physiological functions. Laboratory studies have tested a spectrum of EM fields for bioeffects at cell and molecular levels, focusing on exposures at athermal levels. A clear emergent conclusion is that many observed interactions are not based on** tissue heating. Modulation of cell surface chemical events by weak EM

fields indicates a major amplification of initial weak triggers associated with the binding of hormones, antibodies, and neurotransmitters to their specific binding sites. Calcium ions play a key role in this amplification. These studies support new concepts of communication between cells across barriers of cell membranes; and point with increasing certainty to an essential physical organisation in living matter, at a far finer level than the structural and functional image defined by the chemistry of molecules. New collaborations between physical and biological scientists define common goals, seeking solutions to the physical nature of matter through a strong focus on biological matter. The evidence indicates mediation by highly nonlinear, nonequilibrium processes at critical steps in signal coupling across cell membranes. There is increasing evidence that these events relate to quantum states and resonant responses in biomolecular systems, and not to equilibrium thermodynamics associated with thermal energy exchanges and tissue heating."

7. Free Radicals

7.1 Introduction:

A free radical is an extremely reactive molecule which carries an unpaired electron and which has a very short half-life of 10^{-5} s or less. Although superoxide anions (O_2^-) are the primary oxygen radicals produced in biological systems, they can also give rise to a cascade of other radicals such as hydroxyl, carbonate and lipoperoxy radicals.

Medical literature documents the role of free radicals in carcinogenesis, Guyton and Kensler (1993):

"Cancer in humans and animals is a multistep disease process. In this process, a single cell can develop from an otherwise normal tissue into a malignancy that can eventually destroy the organism. The complex series of cellular and molecular changes that occur through the development of cancers can be mediated by a diversity of endogenous and environmental stimuli. Active oxygen species and other free radicals have been known to be mutagenic; further these agents have more recently emerged as mediators of other phenotypic and genotypic changes that lead from mutation to neoplasia. Free radical production is ubiquitous in all respiring organisms, and is enhanced in many disease states. Free radicals may therefore contribute widely to cancer development in humans."

7.2 Cumulative effects:

Commonly used chemicals and drugs produce damaging levels of free radicals, which produce chromosome and DNA damage and suppress the immune system. Enwonwu and Meeks (1995) review the free radical chemistry of tobacco and alcohol in relation to oral cancer. The abstract is included here to illustrate the central role of free radicals in cancer and immune system suppression, whether they are produced by chemicals, ionizing radiation or non-ionising electromagnetic radiation. They also address the role of free radical scavengers, such as anti-oxidants.

"Abstract :

As shown in this report, abuse of alcohol and tobacco has serious nutritional implications for the host, and generates increased production of reactive free radicals as well as eliciting immunosuppression. Maintenance of optimal competence of the immune system is critical for cancer surveillance. Active oxygen species and other reactive free radicals mediate phenotypic and genotypic alterations that lead from mutation to neoplasia. Consequently, the most widely used chemopreventive agents against oral cancer (e.g., vitamins A, E, C, and beta-carotene) are anti-oxidants/free radical scavengers. These anti-oxidants, both natural and synthetic, neutralize metabolic products (including reactive oxygen species), interfere with activation of procarcinogens, prevent binding of carcinogens to DNA, inhibit chromosome aberrations, restrain replication of the transformed cell, suppress actions of cancer promoters, and may even induce regression of precancerous oral lesions such as leukoplakia and erythroplakia.

7.3 EMR effects on free radicals:

Modulated EMR has been shown to reduce melatonin levels and to lead to an increase in free radicals and increased cell death.

Barnett (1994) reviews possible mechanisms relating microwave exposure to the action of free radicals:

> **"There is increasing support for the theory that free radicals play an important role in discrete, important sub-cellular events during exposure to microwaves. The field of magneto-chemistry is beginning to have an impact on the understanding of subtle effects in molecular biology of cell systems. Chemical bonds consist of paired electrons with opposite spins. Free radicals are highly charges and can only form bonds between radicals of opposite spins. Electron spins may be altered by EM fields and radicals prevented from uniting. Recent information on the small unstable molecule, nitric oxide (NO), as a physiological mediator has shown the importance of oxygen free radicals in biological systems. NO is understood to modulate neurotransmission and regulate cerebral arterial blood flow and has been implicated in the pathogenicity of Alzheimer's Disease.**
>
> **Microwave-induced lowering of phase transition temperature and increasing membrane permeability is inhibited by the presence of antioxidants, thereby implicating free radical involvement. A number of laboratories have reported enhanced permeability to sodium cation in erythrocytes during exposure to microwave fields."**

Adey (1993) discusses McLauchlan (1992) which proposes a model for the production of free radicals by ELF fields. McLauchlan concludes from his model that "the effect begins at the lowest applied field strength, even at levels below thermal noise (kT). The all-important interaction has an energy very much less than the thermal energy and is effective exclusively through its influence on the dynamics; this is counter intuitive to most

scientists." Adey (1993) goes on to consider the work of Grundler and Keilmann (1978) and Grundler and Kaiser (1992) in which around 42 GHz they found highly tunes resonances in yeast cells, with clear responses down to 5 picowatt/cm^2.

Grundler et al. (1992) present a synthesis of interaction of nonthermal EM fields with cellular systems. They present a model of EM field transductive coupling, based on magnetic field-dependent chemical reactions, including cytochrome-catalyzed reactions that involve free radicals, such as reactive oxygen or nitric oxide, leading to a further highly cooperative amplification step. They conclude that in such a system "imposed fields can be active even at intensities near zero."

Lai and Singh, pers. comm. have exposed rat brains to sub-thermal pulsed microwaves (2.45 GHz) and found enhanced single- and double-strand DNA breakage in the presence of enhanced free radicals and accelerated cell death.

The models are now being confirmed by experiments in living tissue as laboratory techniques allow detection of cellular level effects. It is highly likely that the many examples of observed chromosome damage in the presence of RF/MW fields is due to the involvement of free radical mechanisms and/or disruption to intra- and inter-cellular communication. The consequence of this is impairment of the immune system and increased risk of cancer and birth defects, for example.

8. Carcinogenesis processes:

8.1 Introduction:

It has been estimated that 75-80% of all human cancers are environmentally induced, Clemens (1991), 30-40 % of them by diet. The remaining cancers, 35-50%, are primarily from environmental toxins, among which epidemiological research strongly implicates electromagnetic radiation.

Two distinct types of process which lead to neoplasm of cells which can lead to malignant cancer can involve electromagnetic radiation, signal transduction alteration and genetic damage. The first involves the change in signal transduction process in the cells which controls the cells development, and involves calcium ions and/or ODC for example, Byus (1994), Luben (1995), and Weinstein (1991). The second involves DNA and chromosome damage through the action of such agents as free radicals.

A multi-stage process for developing cancer is often described. This starts with initiation, then promotion and finally progression, Weinstein (1988). Adey (1992b) adds "synergism" to include the effects of co-carcinogens.

Initiation involves a single exposure to a carcinogen which damages the nuclear DNA. A single agent (a complete carcinogen) or two or more agents may be necessary, working together in the proper sequence. Promotion involves multiple exposures at certain intervals to agents which do not damage DNA directly. Promotion leads to conversion from benign to malignant tumours. Progression involves the increasing degree of malignancy.

The latency period for most cancers (the time between initiation and appearance of the disease) is often 20 years or more. Initiation is generally thought to change the cell's genetic stores of DNA, but the change is not expressed and a tumour does not result unless one or more promoting agents act repeatedly at a later time. Initiated cells may remain quiescent if they are not stimulated by a promoter, and cancer may never develop if sufficient exposures to promoters do not occur.

In a specific context, tobacco proteins are both initiators and promoters. Because of cigarette smoking's promotional attributes, risks of lung cancer decline after a smoker rejects the habit.

Promotion and progression agents have very weak or no carcinogenic activity when tested alone, but they markedly enhance tumour yield when applied repeatedly following a low or sub-optimal dose of a carcinogen. Promoting agents are not mutagenic and thus are not cancer initiators by an action on the DNA in the nucleus.

Many papers give evidence of EMR as a cancer promoter, e.g. Adey (1992b). Agents which disrupt the gap-junction communication or alter the signal transduction in order to increase proliferation, can be cancer promoters. EMR does change these cellular processes in the same way that known cancer promoters do. In cell cultures the ability of T-lymphocytes (T-cells) to destroy tumour cells is shown, pointing to the importance of the immune system in reducing and eliminating cancer cells. Both 60 Hz ELF and modulated RF fields (450 MHz) fields, Lyle et al. (1983), reduce the lymphocyte killing ability.

Synergism is another form of interaction which occurs when two or more substances potentiate each other's actions, producing more cancers than can be accounted for by the separate effects of each. The phorbol ester TPA is known to activate the membrane bound enzyme protein kinase C (PKC). Studies of these interactions show that PKC plays a critical role in signal transduction in normal cells and it is irreversibly activated by phorbol esters, Adey (1992b). PKC belongs to a group of cAMP-dependent protein kinases identified as sensitive to weak RF fields amplitude-modulated at ELF frequencies, Byus et al. (1984). Many experiments in cell-lines and in animals have shown synergistic effects of EMR and chemical cancer promoters, benzpyrene or TPA for example.

However, evidence is growing that ELF modulated RF/MW radiation not only alters the cellular level growth regulation processes in a cancer promoting way, but also is involved under some circumstances in the breakage of nuclear DNA. Hence EMR appears to be both a cancer initiator and a cancer promoter, which also enhances progression. In this way the similarity with cigarettes is quite strong, as are the similarities to the effects of ionizing radiation, but at a lower, but not insignificant level of impact, particularly because of the near universal exposure of people to RF/MW radiation.

8.2 DNA breakage and Chromosome aberrations (CA) by EMR:

Carcinogenesis can be initiated through breakage of DNA which leads to the aberration of chromosomes. This can happen by the direct action of free radicals or by the inactivation of tumour suppressor genes. Thus it is generally accepted that chromosomal mutations are causal event in the development of neoplasia, Hagmar et al. (1994). Hence, at the population level, an increased frequency of CA has thus been generally considered

indicative of increased cancer risk for those exposed to the damage-inducing agent. Thus it is important to review research which shows CA under EMR exposure.

Two of the important agents identified in these processes are melatonin and free radicals, Liburdy et al. (1993), Reiter (1994) and possibly also calcium ions. CA may be enhanced directly by physiological responses to EMR which reduce the production of melatonin or indirectly by substances such as cysteamine which enhance free radicals. This effect was found in by Kondo et al. (1985) when investigating DNA damage observed after exposure to 1.2 MHz infrasound. Alternatively they may impair the DNA repair mechanism, by altering the cell cycle for example. In either case the result is damaged nucleus DNA.

8.3 Early Biomedical Result: RF breaks chromosomes.

Nature, in March 28th, 1959 contains a paper in the Genetics section entitled "A New Physical Method of creating Chromosome Aberrations". The authors, Drs Heller and Teixeira-Pinto at the New England Institute for Medical Research, report a method they use to prepare medical samples which contain high levels of chromosome aberrations. They use a radiofrequency source of 27 MHz, a pulse length of about 50μs and between 80 and 180 pulses per second (pps).

They report asymmetrical particles aligning themselves along the field lines. They observe that motile bacteria or protozoa migrate along field lines when the RF is on, but resume random movement when the field is turned off. This can be repeated as often as desired. They note that the thermal component is so low as not to affect the viability of these organisms or of mammalian cells. In the larger organisms, they were able to observe intracellular orientation of the subcellular particles. They say that this led them to believe that this force might be used as a powerful and controlled mutagenic agent.

Growing garlic roots were exposed to the field and the water they were in was monitored and no temperature rise was seen. The tips were exposed to the RF field for 5 mins and examined 24 h later. They observe, Heller and Teixeira-Pinto (1959):

> **"Among those aberrations seen were linear shortening of chromosomes, pseudochiasmata, amitotic division, bridging, irregularities in the chromosomal envelope. The effects noted mimic those produced by ionizing radiation and c-mitotic substances."**

Of the papers and reviews I have, it is only cited in Shore (1981), the WHO review "Environmental Health Criteria 16: Radiofrequency and Microwaves" This short paper is remarkable for its significance and the fact that it is been almost totally ignored by subsequent reviews and reports. The conclusion about the similarity of effects to those of ionizing radiation and other cell damaging agents is telling. It also related to the role of free radicals, which are known to be produced and cause DNA damage under exposure to ionizing radiation and have been observed in vivo under microwave exposure, Lai and Singh pers. comm. It is also noted in a mouse reproductive study by Dimberg (1995), who used 20 kHz magnetic field with a peak-to-peak amplitude of 15 μT (sawtooth wave). He concludes: "Most of the effects of MF (magnetic field) treatment during the embryonic period were similar to those induced by ionizing radiation but much weaker".

8.4 ELF studies involving chromosome aberrations, DNA breakage and cancer:

Several ELF exposure studies have been carried out on workers which are of relevance because to the strong similarity between effects of ELF EMR and ELF modulated RF/ME EMR.

Murphy et al. (1993) note that since epidemiologic studies have reported a modestly increased risk of childhood leukemia associated with certain electric power wire configurations and since cancer it likely to involve DNA damage, this review discusses the evidence of direct and indirect genetic toxicity effects for both electric and magnetic fields at 50- and 60-Hz and miscellaneous pulsed exposures. Exposure conditions vary greatly among different end points measured, making comparisons and conclusions among experiments difficult. Although most of the available evidence does not suggest that electric and/or magnetic fields cause DNA damage, the existence of some positive findings and limitations in the set of studies carried out suggest a need for additional work. Additional work has been done.

Ciccone et al. (1993) conducted a case control study of 50 acute myeloid leukemias (AML), 17 chronic myeloid leukemias (CML), 19 myelodysplastic syndromes (MDS), and 246 controls. The chromosome aberrations were recorded according to the International System for Human Cytogenetic Nomenclature. Chromosome aberrations were not associated with chemical exposures (OR = 1.0), but a non-statistically significant excess was noted in association with electromagnetic fields (OR = 2.1).

Valjus et al. (1993) sampled for chromosomal aberrations, sister chromatid exchanges (SCEs), replication indices and micronuclei in peripheral blood lymphocytes among 27 nonsmoking power linesmen with considerable long-term exposure to 50-Hz EM fields, and among 27 nonsmoking telephone linesmen serving as a reference group, pairwise matched with the exposed workers for age and geographical region. Blood samples from the two groups were collected, cultured and analysed in parallel. No differences between the groups were observed on analysis of SCEs, replication indices or micronuclei. However, the mean rate of lymphocytes with chromatid-type breaks was higher among the power linesmen (0.96% gaps excluded, 1.41% gaps included) than among the reference group (0.44% and 0.70%, respectively). The excess of aberrant cells was concentrated among those power linesmen who had worked earlier in their life. Although the interpretation is somewhat complicated by the confounding effect of previous smoking, these results suggest that exposure to 50-Hz EM fields is associated with a slight increase in chromatid breaks.

Skyberg et al. (1993) studied 13 high-voltage laboratory employees and 20 referents participated in a cross-sectional, matched-pairs study of cytogenetic damage. During cable testing the workers were exposed to static, alternating, or pulsed electric and magnetic fields. The alternating magnetic field levels of 50 Hz were 5-10 μT, occasionally much higher. Chromosome aberrations, sister chromatid exchanges, and aneuploidy were studied in peripheral blood lymphocytes. Among seven smoking laboratory employees the mean number of chromosome breaks/200 cells was 2.3, as compared with 0.7 for the job-matched referents. The comparable figures for inhibited cultures were 12.0 versus 6.0. No increase was detected in nonsmokers with either method. The results support, to some extent, the hypothesis of an increased risk of genotoxic effects among high-voltage laboratory workers, particularly a synergistic effect with smoking.

Nordenson et al. (1994) reported that their recent studies have shown a significant increase in the frequency of chromosomal aberrations in human amniotic cells after exposure to a sinusoidal 50 Hz, 30 μT (rms) magnetic field. To evaluate further interactions between chromosomes and electromagnetic fields, they analyzed the effects of intermittent exposure. Amniotic cells were exposed for 72 h to a 50 Hz, 30 μT (rms) magnetic field in a 15 s on and 15 s off fashion.

Eight experiments with cells from different fetuses were performed. The results show a 4% mean frequency of aberrations among exposed cells compared to 2% in sham-exposed cells. The difference is statistically significant, with $P < 0.05$ both excluding and including gaps. In another series of eight experiments, the cells were exposed in the same way but with the field on for 2 s and off for 20 s. Also in these experiments a similar increase in the frequency of chromosomal aberrations was seen, but only when the analysis included gaps. Continuous exposure for 72 h to 300 μT, 50 Hz, did not increase the frequency of chromosomal aberrations.

8.5 RF/MW studies:

Garson et al. (1991) studied 38 Australia Telecom radio-linesmen who had been exposed to RF EMR in their work and compared the chromosome damage in lymphocytes compared 38 non-exposed clerical staff. A very detailed assay of chromatid and chromosome gaps and breaks and other aberrations was carried out. Most categories showed a small but statistically insignificant increase in chromosome aberrations, with the sum of aberrations of 2.55% for linemen and 2.18% for controls (95%CI: 0.9-1.6).

For Chromatid Gaps RR=1.2 (0.7-2.1); Chromosome Gaps: RR = 1.5 (0.6-3.5); and Chromosome Breaks (without outlier) 1.4 (0.8-2.3). Adjusting for confounding from recent X-rays and for smoking both produced a small increase in Rate Ratio. The absence of adjusting for coffee drinking is a limitation. Such an adjustment would be likely to favour reduction in the incidence among clerical workers, further increasing the Rate Ratio. The incidence of total chromosome aberrations among the controls does appear rather high.

Hagmar et al. (1994) trichotomize CA into the low (1-33%ile), medium (34-66%ile) and high (67-100%ile). The threshold for low CA is typically 1.0% but in the range 0.5 to 1.5 %, while medium is typically 1.0 to 2.0 %, and high >2 %, but may use a threshold between medium and high of 3 %. Taking the typical classification the Australia Telecom study gas both exposed and control groups in the high category. If the control group was in the "low" category <= 1%, then the Rate Ratio for the clerical staff would be 2.2 and for the linesmen 2.6, both of which are significant ($p<0.01$).

Timchenko and Ianchevskaia (1995) concluded that an electromagnetic field (EMF) at a frequency of 24 or 14 MHz and intensity of 400 or 200 V/m, increases numbers of epatocytes from rats with chromosomal aberrations 1.4-1.5-fold.

8.6 DNA breakage associated with RF/MW exposure:

Sagripanti and Swicord (1986) showed that non-thermal levels of microwave exposure can produce single and double-strand DNA breaks in E. coli in solution.

Garaj-Vrhovac et al. (1991) showed that cultured V79 Chinese Hamster fibroblast cell exposed to continuous wave (CW) 7.7 GHz microwaves at power density of 0.5 mW/cm^2 for 15, 30 and 60 min produced a significantly high frequency of specific chromosome aberrations such as dicentric and ring chromosomes in irradiated cells. The dose-response relationships were significant at $p<0.01$.

Garaj-Vrhovac et al. (1992) exposed whole human blood samples to the same exposure regime. with the addition of power densities of 10 and 30 mW/cm^2. The number of chromosome aberrations increased from 1.5 % in controls to 2.7 to 7.2 % at the rising power densities. There was a statistically significant dose response with $p<0.05$ for total aberrations, $p<0.001$ for Accentric and $p<0.0001$ for micronuclei.

Sarkar et al. (1994) found significant modification of the DNA from mouse cells from brain and testes exposed to 1 mW/cm^2 2.45 GHz microwaves for 2 hr/day for 120, 150 and 200 days.

Lai and Singh (1995) exposed living rats brains to a single 2 h exposure to microwaves at 2.45 GHz, pulsed at 500 pps, at SARs of 0, 0.6 and 1.2 W/kg. They found significant dose-response relationships for single strand DNA breaks in an assay carried out 4 hours after exposure for both the hippocampus and the rest of the brain. A second analysis involved assaying the whole brain and continuous wave microwaves at 2.45 GHz and 1.2 W/kg. This showed a statistically significant increase in single-strand DNA breaks between sham and exposed ($p<0.01$) but no significant difference between assays at 0 h and 4 h after exposure.

Lai and Singh (1996) repeated the experiment of Lai and Singh (1995) and extended the analysis to include and assay of double-strand DNA breaks and included both pulsed (500 pps) and continuous microwaves at 2.45 GHz. The exposed condition was 2mW/cm^2 (SAR = 1.2 W/kg). Statistically significant single-strand DNA breaks were found for both the CW and pulsed signals ($p<0.01$), and for double-strand DNA breaks (pulsed $p<0.01$ and CW $p<0.05$). This data was not available for the MacIntyre Case.

Their most recent work, Prof H. Lai, pers. comm., that in the exposed rats brains there is enhancement of free radicals and the acceleration of cell death (apoptosis). It is not yet known whether this is caused by the MW radiation influencing the pineal gland or the retina of the eyes, to reduce melatonin production and hence enhance free radical numbers, or whether the MW radiation produces free radicals locally in the brain.

This is relevant to the study carried out by Adey et al. (1996) in which rats exposed to cellphone-like signals had 30 % fewer tumours than controls and the tumours were statistically significantly smaller. These results were reported to the 1996 BEMS conference in Victoria BC. Dr Singh raised the question with Dr Adey, of the possibility of cell death as an explanation for the result. Dr Adey agreed that this was possible, but stated that it needed to be tested. Lai and Singh have found that result.

Hence it has been shown that a sub-thermal dose of microwaves (0.6 W/kg and 1.2 W/kg) can enhance DNA breakage and accelerate the cell death in living brains, through the increased production of free radicals, at such a rate that tumour cells die at a faster rate than they grow, producing fewer and smaller tumours.

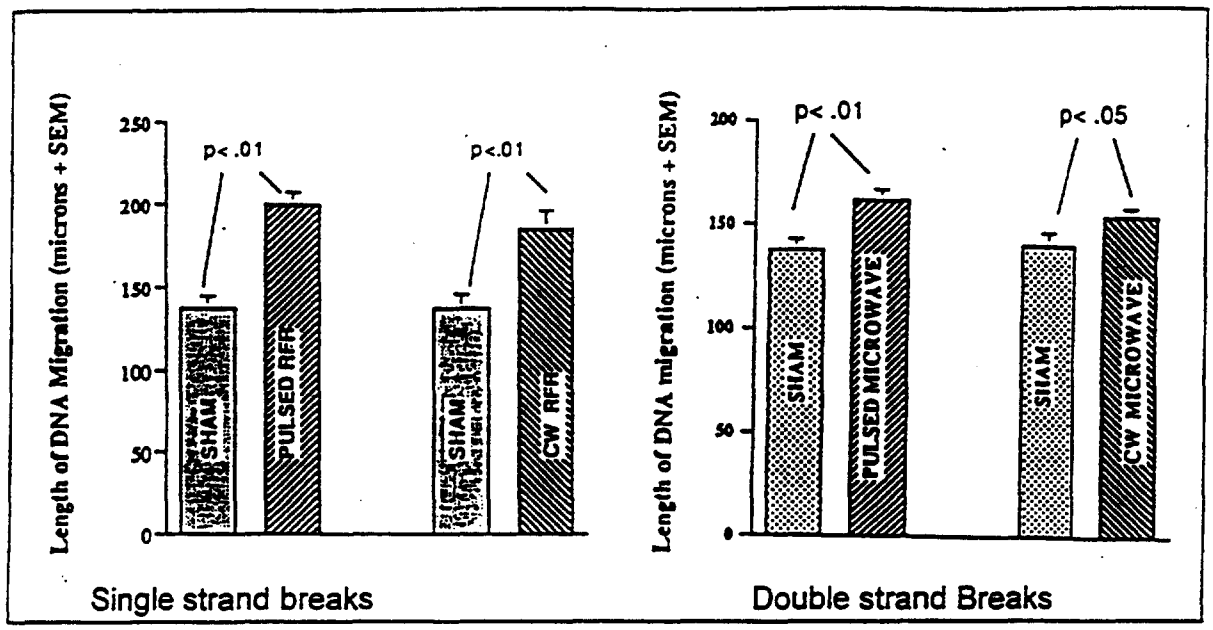

Figure 19: DNA breakage in rat brains (SAR = 1.2 W/kg), Lai and Singh (1996).

All of these above experiments were carried out without the use of cancer initiators nor co-carcinogens. They involve the direct application of RF/MW radiation to a sample or an animal and the observation of chromosome breakage, DNA breakage, tumours, free radicals and cell death. Hence they confirm the proposal of Reiter (1994) in section 3.1, that EMR would be both an initiator and promoter of cancer, in his case through melatonin reduction, in this case through direct observation of DNA damage which might involve melatonin reduction since free radicals are observed to be enhanced.

8.7 Cellular Base Station radiation's synergistic mutagenic effect with MMC:

A Belgian research team has found that "very-close-range" exposure to microwaves from a cellular telephone base station increases the effect of a chemical mutagen on human blood cells, Maes et al. (1996). Whole blood samples were exposed to 954 MHz microwaves from an actual GSM base station and then to the DNA damaging agent mitomycin C (MMC). Other samples were exposed to either microwaves or MMC alone.

The exposure was at 5 cm from a GSM digital 15 W antenna, giving an SAR of 1.5 W/kg, for a period of 2 h. At 954 MHz, σ= 1 S/m, and hence S = 796 $\mu W/cm^2$, from Eq. 11. This is a high, but significantly non-thermal exposure.

In this experiment, base station levels of microwaves alone showed no significant mutagenic effects. However, blood samples exposed to microwaves and then to MMC showed a considerably higher, statistically significant number of chromosomal abnormalities than those exposed to MMC alone. Microwave exposure increased the subsequent effect of MMC by about 20 to 50 %, the higher levels being produced by higher concentrations of MMC.

It is important to determine what the dose-response relationship of this exposure is. Clearly a non-thermal mechanism is operating, as will many other chromosome aberration

observations reported here. These results show that GSM digital microwave radiation is co-carginogenic with other natural or environmental carcinogens.

Thus people who are exposed to GSM bases station microwaves have a higher risk of cancer and reproductive effects by making chemical carcinogens more potent in damaging chromosomes. A potentizing effect with skin cancer and UV is a possibility.

On the other hand, the research of Lai and Singh shows that the microwave exposure levels produced by cellular telephones in users heads, free radical production is enhanced, breaking DNA and enhancing the rate of cell death in the brain. The Belgian research also suggests that the whole body exposure of the cellular telephone antenna can enhance the risk of chemical damage to chromosomes. This might involve liver cancer for cell phones hooked onto users belts, or lung cancer and breast cancer when the cell phone is kept in a breast pocket.

8.8 Conclusions on Mutagenic effects of EMR:

Chromosome Aberrations and DNA damage has been found under non-thermal exposure to EMR. ELF and ELF modulated RF have been associated with chromosome aberration in cells and in exposed workers. Microwaves have been shown to produce DNA damage in living rats brains. Microwaves have also been shown to potentize cancer initiators (MMC) and to enhance the chromosome aberrations with exposure to a GSM digital base-station near field signal. Hence EMR is implicated in increasing cancer rates in exposed populations, Hagmar et al. (1994).

Increased cancer incidence can come about by the direct effect of a DNA damaging carcinogen or by the synergistic effect of co-carcinogens. The co-carcinogenistic effect and cancer promotional effect of EMR has been widely suggested and demonstrated through a number of experiments, e.g. Adey (1992b), Byus et al. (1988). Direct effects (in the absence of a cancer initiator) include chromosome aberrations and DNA breakage which is most likely to be the result of the enhanced presence of free radicals in the RF/MW field. The role of melatonin is important here. Direct effects are likely to involve higher mean power densities than co-carcinogenetic effects. In Lai and Singh (1995) the inter-animal variability is very small giving a small standard deviation for each exposure group. Even so a linear the dose-response relationship is statistically significant for the "rest of the brain" assayed 4 h after exposure ceased. This suggests that the smallest detectable increase in DNA breakage would be associated, with this small sample size, with an SAR of <0.2 W/kg , σ = 1.7, S < 62μW/cm^2. No clear lower limit is able to be estimated.

9. Long-term Animal Studies:

Very few long-term animal studies involving RF/MW exposure have been carried out, largely because of their extreme difficulty and very high cost. The significant studies, also reviewed by the U.S. E.P.A., are reported here.

9.1 University of California, Berkeley:

Professor Charles Susskind and Dr Susan Prausnitz, Dept of Electrical Engineering, UC Berkeley carried out the first reported long term study for the US Air Force. Prausnitz and

Susskind (1962). They exposed male Swiss albino mice to 9.27 GHz microwaves, pulsed with a 2 μs pulse at 500 Hz, 4.5 mins per day, 5 days per week for 59 weeks with an exposure level of 100 $\mu W/cm^2$. This amounts to a mean weekly exposure of 0.22$\mu W/cm^2$.

Detailed autopsies were carried out on 60 irradiated and 40 control mice who died during the experiment. Two adverse effects were more severe in the exposed compared to the control animals.

(1) Testicular degeneration (atrophy with no sperm) occurred in 40 % (23/57) of the exposed animals and 8.1 % (3/37) of the control animals.

(2) Cancer of the white cells or leucosis was seen in 35 % (21/60) of the exposed animals compared to 10 % (4/40) of the controls. This condition was described as monocytic or lymphatic organ tumours or myeloid leukaemia in the circulating blood.

At the 16-month interim kill, one month after exposure ceased, 30 % (6/20) if the exposed group had leucosis compared to 10 % (1/10) of the controls.

At the final kill at 19-months, 4 months after cessation of exposure testicular atrophy was seen in 21% (14/67) of the exposed group and 5 % (1/19) of the control group, and testicular weights were lower for the exposed group. At this stage leucosis was the same in both groups at 18 % (12/67) for the exposed group and 21 % (4/19) for the control group.

This gives an overall rate for testicular degeneration of 29.8% (39/124) for the exposed group and 7.1% (4/56) for the control group, giving a Rate Ratio of RR=4.2 . For leucosis the incidence was 26.5 % (39/147) for the exposed mice and 13.0% (9/69) for the control mice, RR = 2.04 .

These combinations of symptoms pose some challenging interpretations. Testicular degeneration is not associated with the brief heating effect of the daily exposure (4.5 mins at 100$\mu W/cm^2$), because this is usually taken to be a non-thermal exposure. The current NZ standard for public exposure for microwaves is 200$\mu W/cm^2$ and there is a proposal to relax it to 1000$\mu W/cm^2$, which is also claimed by those who believe that only thermal effects exists, to be harmless and non-thermal. In addition, Caimie et al. (1980) exposed mice to microwaves at power density of 50 mW/cm^2. They found that the absorbance in the abdomen area of the liver was 11 times greater than the testes, and while the abdomen temperature was increased the testicular temperature was not.

Leucosis requires damaged DNA and chromosome aberrations which are transferred from cell to cell through mutation. The same mechanism could cause testicular degeneration. An accumulated cellular level damage mechanism is not necessarily related to the intensity but can relate to total dose in relation to rates of repair. Hence the averaging of weekly exposure is a meaningful adverse effect related level. Actual public exposure levels of 0.2$\mu W/cm^2$ and less saw childhood leukaemia incidence and death rate rises of similar values (2.74 for mortality) in the North Sydney Study.

9.2 University of Washington Case Study:

Establishment of a potential adverse human health effect can be obtained from a suitably designed and executed animal experiment. Such an experiment was carried out at the University of Washington by Professor Arthur Guy and his associates, funded by the United State Air Force. The exposed a large group of rats to pulsed radar-like microwaves, at 0.4 W/kg, the human exposure level allowable under the ANSI standard. These rate were compared to a similar group who were sham exposed.

Guy et al. (1985) reported that there was no statistically significant difference in tumours between the two groups except some benign adrenal tumors, which occurred earlier in the exposed animals. The reported result is so equivocal that it does not rate any further mention than this in the Repacholi (1993) review. On the other hand, the EPA review team worked with the original University of Washington research team, and undertook further statistical analysis of their results and showed "a statistically significant elevation in the incidence of carcinomas but not sarcomas, at all sites combined."

(Carcinoma: a malignant new growth made up of epithelial cells tending to infiltrate the surrounding tissues and give rise to metastases.

Sarcoma: any of a group of tumours usually arising from connective tissue, although the term now includes some of epithelial origin.

Epithelial: :Pertaining to the internal or external surfaces of bodies, including the lining of vessels and other small cavities.

Metastases: The transfer of disease from one organ or part of an organ to another.)

The experiment ran for 25 months with some mice being sacrificed and analysed at 13 months. Their initial reports concluded no effects except a significant increase in the number of benign adrenal tumours. At 13 months the exposed group had a significantly larger number of B- and T-cells than do controls, but no difference was seen at the end of 25 months. This suggests the immune system was initially disrupted, but over a 2 year period it adapted to the exposure situation. Disturbance of the immune system is also consistent with the developing cancer and tumours growth.

These results were worrying to EPA researchers. Dr Robert McGaughy asked Dr Lawrence Kunz, the pathologist at the University of Washington, for copies of the survival and histropathologic findings. These results are listed in Table 3.

Separating these tumours into carcinomas and sarcomas, Dr McGaughy was able to show that three statistical tests showed a statistically significant increase in carcinomas ($P<0.05$) but no statistically significant increase in sarcomas. The EPA team argue that while most chemical carcinogens affect only one or a few tissues, the distribution of the EM field as a "toxic agent" is more uniform than a "typical" chemical agent, and therefore an "all sites" approach is justified. This is sound since incident RF/MW fields induce electric field gradient changes as the cellular level over the whole body, thereby changing the pineal and serum melatonin, altering the gap-junction potential, changing cellular membrane permeability, altering the signal transduction process, the calcium ion efflux and generating free radicals.

Table 3: Crude incidence of neoplastic lesions (Tumours)

Site/Type	Crude Tumor Incidence			
	Control		Exposed	
Adrenal Cortex	12/85	14.1 %	12/76	15.8 %
Adrenal medulla	1/73	1.4 %	7/67	10.4 %
Thyroid	9/85	10.6 %	12/76	15.8 %
Liver	1/85	1.2 %	3/76	3.9%
Pituitary	21/85	24.7 %	19/75	25.3 %
Testes	0/85	0 %	2/76	2.6 %
Epididymis	0/85	0 %	1/76	1.3 %
Pancreas	2/85	2.4 %	2/76	2.6 %
Urinary bladder	0/85	0 %	2/76	2.6 %
Stomach	4/85	4.7 %	4/76	5.3 %
Duodenum	0/85	0 %	1/76	1.3 %
Lymph node	0/85	0%	1/76	1.3 %
Soft Tissues, Thorax	0/85	0 %	2/76	2.6 %
Mesentery	0/85	0 %	2/76	2.6 %
Lymphosarcoma	3/85	3.5 %	4/76	5.3 %
Total	53/85	62.4 %	63/75	84.0% (RR=1.35,p<0.05)

McGaughy et al. (1990) point to the more ubiquitous action of melatonin as an example, since,

"Nocturnal pineal melatonin activity is known to be inhibited by ELF electric fields (Wilson et al 1986) and that the pineal gland function is closely coupled to the function of other glands. Melatonin is known to inhibit tumour growth-enhancing hormones like prolactin and estrogen. The postulate has been made that when the blood melatonin concentration decreases because of the action of EM fields on the pineal gland, a tumour growth inhibitor has been reduce or effectively removed, thereby causing a stimulation of tumour growth.

Although only breast and prostate tumours have been discussed in this connection, the same regulation by melatonin might hold for other hormonally-regulated endocrine organs as well."

The Guy et al. (1985) study, along with other supporting material, led to the recommendation that the US EPA classify RF/MW as a possible human carcinogen (Class C).

The data presented in this report indicate the progressively strengthening evidence of carcinogenicity and other adverse health effects from chronic non-thermal exposure to RF/MW radiation which raise the evidence to classify RF/MW radiation as a highly probable (Class B1) carcinogen.

Note: All you need in New Zealand Law is evidence of a potential irreversible adverse environmental effect to decline this application and to recommend the identification of a site in a less sensitive receiving environment, or a potential adverse effect to require mitigation or remediation.

9.3 Polish Study:

Szmigielski et al. (1982) measured the effects of 2.45 GHz microwave radiation at 5, 10 and/or 15 mW/cm^2, 2h /day, 6 times/week exposure (average weekly exposure 0.36, 0.52 and 1.1 mW/cm^2), mice able to maintain core temperature under both exposures, specifically investigating lung cancer, breast cancer and skin cancer. Figure 20 shows the result of initiating skin tumours using 3,4 benzo-alpha-pyrene (BP) and assessing the cancer promoting effect of microwaves.

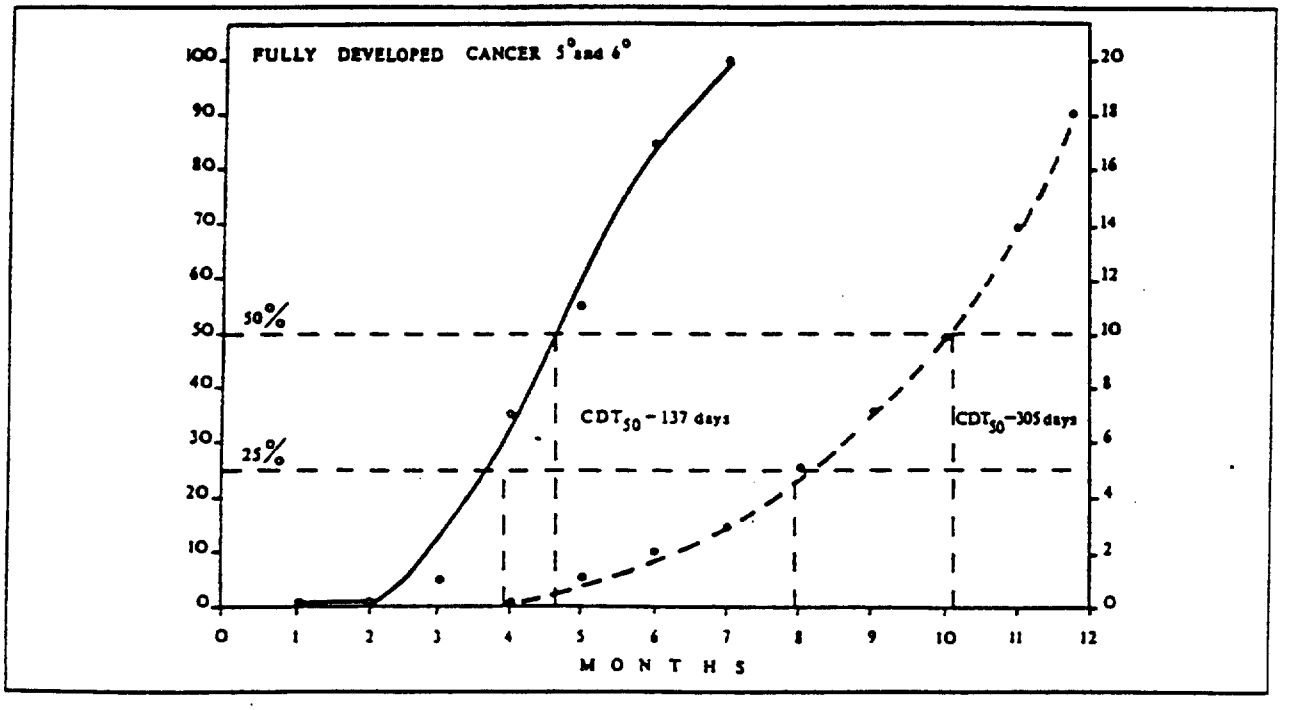

Figure 20: Growth curves of BP-induced skin tumour in mice exposed daily to 10 mW/cm^2 of 2.45 GHz microwave radiation for the whole period of tumour growth. CDT_{50} refers to the cancer development time when 50 % of the animals have tumours.

Cancer development started 2 months earlier for the MW exposed mice and reached the 50 % point for the population after 137 days compared to 305 days. Hence MW significantly accelerated the growth and proliferation of skin cancer tumours.

Figure 21 shows the results of planting lung cancer (sarcoma) cells and then exposing the mice to 5 and 15 mW/cm^2 MW radiation. The 5 mW/cm^2 exposure produced an

enhancement of lung cancer nodules at 2.5 times more than controls after 3 months, but at a similar level to the effect of an over-crowding stress factor. The 15 mW/cm^2 exposure produced about 5.5 times more lung cancer nodules.

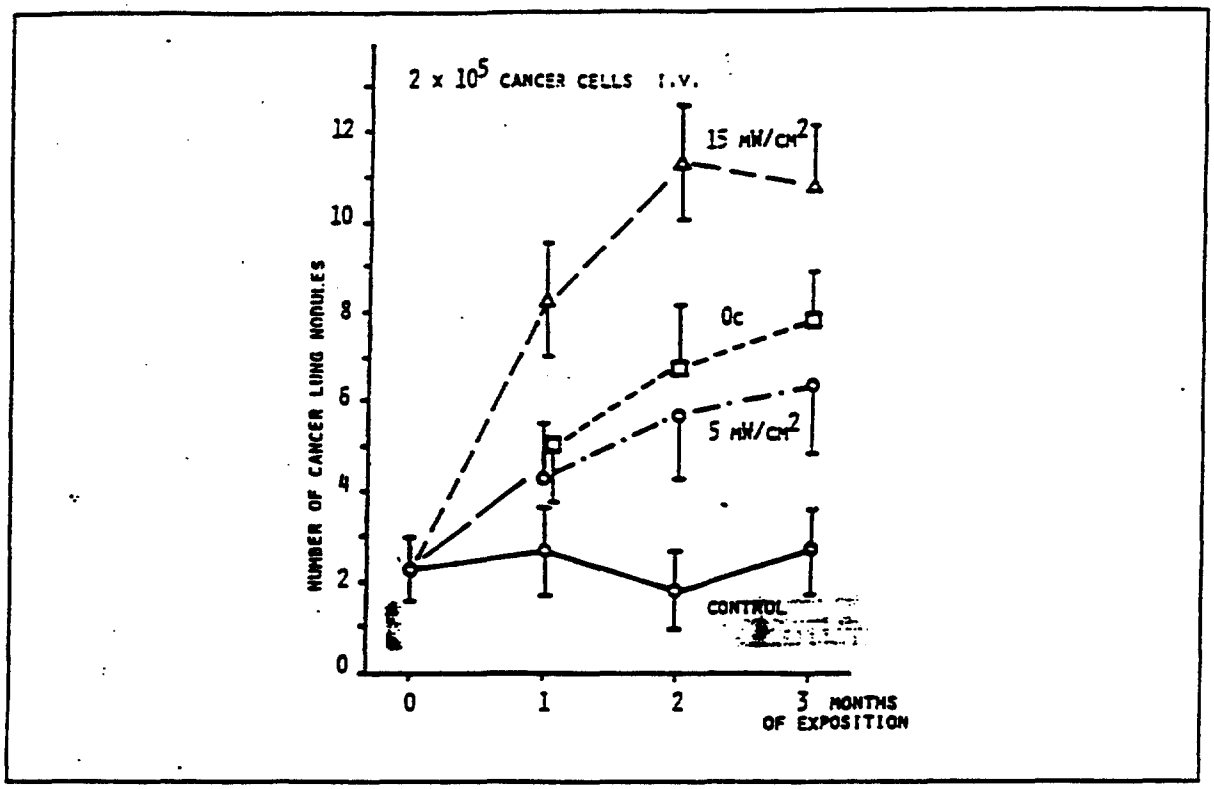

Figure 21: The number of lung tumours (following intravenous injection of 2 x 105 viable sarcoma cells) in mice exposed during 1, 2 and 3 months to 2.45 GHz microwaves (2h daily) at 5 or 15 mW/cm^2. Oc refers to mice treated with nonspecific stress of over crowding.

A parallel experiment for breast cancer for control, overcrowding stress, 5 and 15 mW/cm^2 MW exposure, the 50 % development points were 322, 255, 261 and 219 days, respectively. These show a similar relationship to the results in Figure 13 for lung cancer, except that the stress and 5 mW/cm^2 effects are reversed.

These results show statistically significant increases in numbers and rates of development of chemically initiated skin, lung and breast tumours when exposed to low level microwaves, with a significant dose response relationship in each case.

9.4 Duke University Medical Center:

Eight week old female mice were exposed to 2.45 GHz microwaves at power densities of 5 to 15 mW/cm^2 for 30 min/day over periods between 1 and 17 days, Huang and Mold (1980). Daily mean exposures were about 100 to 300 μW/cm^2, and exposure conditions were essentially isothermal. The results showed, (a) A sustained activation of tissue macrophages resulting in suppression of lymphocyte responsiveness, and (b) a gradual but temporary stimulation directed to the lymphocytes. Macrophage activation may have caused the early depression of lymphocyte responsiveness. The suppression is later

overridden by the cumulative direct stimulation of lymphocytes by microwaves. Prolonged exposures is suggested to eventually result in depressed function in much the same as seen in rheumatoid arthritis which occurs from chronic immune stimulation.

They also conclude that 2.45 GHz microwaves affect the hematopoietic colony-forming abilities through altering the growth of both erythroid and myeloid cells. This is direct evidence of the ability of sub-thermal microwaves to cause chronic immuno-suppression.

9.5 Summary and Conclusions about long-term animal experiments:

Animal experiments confirm that in mice pulsed RF/MW radiation is able to initiate statistically significantly more malignant tumours in many body organs at exposure levels assumed to be non-thermal and safe (0.4 W/kg), McGaughy (1990), and in the presence of a chemical cancer initiator to drastically increase the rate of development of lung, breast and skin cancer, Szmigielski et al. (1982), showing the strong co-promotional effects of microwave exposure. Prausnitz and Susskind (1962) found increased in testicular degeneration and increases in leukaemia at Rate Ratios and mean weekly exposure levels which are compatible with the North Sydney Study. These are consistent with the research summarized above on the direct mutagenic affects of RF/MW radiation and the research showing alteration of signal transduction, cell communication which influence the cellular level growth regulation and can lead to cell proliferation and thence to tumour formation and cancer.

Sub-thermal microwaves caused significant impairment of the immune system functioning.

10. Reproductive effects and Teratology:

10.1 Introduction:

The cellular level changes discussed and documented above are pertinent also to the consideration of potential or actual effects of RF/MW radiation on the development of human embryos, miscarriage and adverse birth outcomes.

Altered signal transduction and gap-junction communication, or DNA breakage and Chromosome Aberration in the developing human embryo is potentially damaging or fatal. Brent et al. (1993) record that 20 to 25 % of human birth defects are caused by genetic factors. Laboratory studies using mice and other animals have typically employed exposure levels in the range 10 to 100 mW/cm^2, or even higher, in the belief that the higher the dose the more likely the change of detecting a result.

The cellular processes discussed above show the fallacy of this, as does the calcium ion windowing effects, which have been monitored to change in association with environmental exposure levels of less than 10 μW/cm^2. When moderate to high exposure levels are used great care must be taken to discern between thermal effects and non-thermal effects. Real non-thermal effects can be masked by large thermal effects. The following are a sample of laboratory experiments involving mice and chickens exposed to microwaves.

10.2 Animal Studies:

Chazan et al. (1983) investigated the development of murine embryos and fetuses after irradiation with 2450 MHz microwaves. They found indications of retardation of development in the early period of gestation in mice exposed to thermal MW fields. In mice exposed to microwaves at 40 mW/cm^2 during the second half of pregnancy increased number of resorptions, stillbirths and internal hemorrhages was noted. The living fetuses had lowered body mass compared to the offsprings of sham-irradiated mice.

Berman, Carter and House (1982) found reduced weight in mice offspring after in utero exposure to 2450-MHz (CW) microwaves. They were exposed to 28 mW/cm^2 for 100 minutes daily from the 6th through 17th day of gestation. The offspring were examined either as fetuses after hysterotomy on the 18th day of gestation or as naturally born neonates on the 1st and 7th day of age. Fetuses of half of the dams were examined on the 18th day of gestation. The incidence of pregnancy and the numbers of live, dead, resorbed, and total fetuses were similar in both groups.

The mean weight was significantly lower (10%) in live microwave-irradiated fetuses, and ossification of sternal centers was significantly delayed. In the offspring that were born naturally, the mean weight of microwave-irradiated 7-day-old suckling mice was significantly lower (10%) than that of the sham-irradiated group. Survival rates of neonates in these two groups were not different. These data demonstrate that the decreased fetal weight seen in microwave-irradiated mice is retained at least 7 days after birth. Evidence from other published studies is presented to show that the retarded growth is persistent and might be interpreted as permanent stunting.

Suvorov et al. (1994) studied the biological action of physical factors in the critical periods of embryogenesis. The critical period in a chicken embryonic development (the 10-13-th days of incubation) is revealed under total electromagnetic radiation. EMR is a physiologically active irritant which can influence functional state of the brain. The increased absorption of electromagnetic energy takes place in this incubation period. Its dynamics within 20 days of embryonic development has phasic, up and down character.

Electromagnetic exposure (4 hours a day) in the above mentioned period evokes a delay in embryo adaptive motor behavior (biofeedback learning). Morphological investigation shows significant pathological changes, specifically, destruction of share brain synapses. The delay in embryo hatching for a day is also detected. Radiation exposure within other periods of incubation (3-6-th or 12-15-th days) was not effective with respect to formation of normal motor pattern in biofeedback experiment. Unfortunately this paper is in Russian and no exposure levels are quoted in the English translation of the abstract.

Prausnitz and Susskind (1962) were not studying reproductive effects, but atrophy of the testes would have severe effects on any sperm which survived. Such sperm are unlikely to have undamaged DNA. Their exposure regime was 100 μW/cm^2 for 4.5 mins/day, averaging 0.22 μW/cm^2 /week.

10.3 Summary and conclusions about teratological animal studies:

The in utero developing embryo is very vulnerable to damage from toxins. At critical times damage to certain organs occurs. With sufficient foetal or placenta damage a spontaneous abortion is initiated. At other levels and timing of damage a still birth can result. Thermal levels of microwave exposure has produced retardation of development if exposure is in early pregnancy, and resorptions, still births and hemorrhages with exposure in the second half of the pregnancy.

A much lower microwave dose was associated with significant reduction in birth weight and permanent stunting and slowing of bone hardening. Changes in chick embryo biofeedback learning is observed and testicular atrophy was observed with a mean exposure to a radar-like signal averaging 0.22 $\mu W/cm^2$ over a week.

RF/MW radiation causes significant birth and reproductive damage in exposed animals at thermal levels and at very low short-term and extremely low average exposure levels.

11. Conclusive evidence of non-thermal effects:

From the beginning, when we considered melatonin and free radicals, right through this review, the mechanisms have been non-thermal. Research studies have documented many non-thermal mechanisms and yet industrial consultants and government officials continue to ignore this massive body of evidence and claim that there are no non-thermal effects of RF/MW radiation. Much of this attitude is sourced back to the early days of U.S. post-war research, the Tri-Services Program, and the pioneering work of Professor Herman Schwan of the Department of Bioengineering, University of Pennsylvania. For example, Schwan and Foster (1980) conclude: "The considerations above do not suggest any weak nonthermal mechanism by which biological systems could react to low-intensity microwave fields."

Dr Alan Frey, Frey (1988), outlines the historical development of research on low intensity nonionizing radiation. He points out that most of the research done during the 1960's and 1970's was irrelevant to his topic since the Department of Defense sponsors who determined what would be done were interested only in high power levels relevant to thermoregulation. This was based to a significant extent on the notions about the nervous system function which Professor Schwan had developed. He set up a mathematical model of tha axon membrane, and assumed that this was a reasonable representation of the nervous system, Schwan (1969). His calculations with the model indicated that at field strengths that are "not thermally significant", the induced potentials across the nerve membrane are many orders of magnitude smaller than the nerve resting potential. He stated that such induced fields applied to the resting potential of the axon cannot excite the nerves, and essentially, on the basis of this, he concluded that the nervous system could not be influenced by low intensity RF radiation.

It is evident that this is in stark contrast to most of the research already presented. How can the differences be resolved ?

Dr Frey points out that he had identified two primary faults in Prof Schwan's model. One was that the model was unrealistic. Nerves function, and the resting potential, is only one extreme of the continuum of potentials of the axon. Schwan ignored most of the nerve

cell, including the most important part, when he considered only the axon in his model. Further, nerves interact (see the dendritic cell in Figure 16). The points of interaction on the synapses are the most sensitive to disturbance, not the axon. Thus Schwan's model, based on the resting potential of the axon, did not conform to reality.

Secondly, Schwam assumed that we had a good undertanding of the nervous system at the time he developed his model. What we have learnt since then shows how complex it is. Cellular biochemisty has identified and quantified many processes in cells, including nerve cells, many of which are altered by very small and subtle EMR induced signals. Frey (1971) showed that by changing one of Schwan's model parameters to a more realistic value, and then doing his calculations, leads to the conclusion that the nervous system would have been affected by RF radiation.

Frey's work in 1971 has been vindicated by many studies but those studies and Frey (1971) have been totally ignored by those favouring a thermal view. More sophisticated models are now in use with considerable experimental verification. These are outlined by Adey (1981, 1991) for example.

To claim that there are no nonthermal mechanisms for the interaction of weak RF/MW signals with human and animal organs and cells is simply not scientifically credible.

Many experiments are deliberately undertaken under non-thermal or isothermal conditions, and many of them show significant cellular, organ or whole animal changes.

Altering the nature and rate of signal transduction processes, including calcium ion fluxes, at the cell level involves subtle but important changes to cell growth and cell death rates which have clear implications for health.

We have seen that EMR changes the fundamental growth regulation factors at the cellular level and that animals chronically exposed to a range of RF/MW regimes have produced benign and malignant tumours, reproductive problems and cancer of the blood. Behaviour is found to change and brain activity alters. Changes in lymphocytes and calcium ions relate to EMR interference with the immune system. The next section assesses whether human epidemiological and behavioural studies find a measurable change in health and well-being under chronic low exposure to RF/MW radiation.

12. Epidemiological Studies:

12.1 North Sydney Study:

12.1.1 Introduction:

Hocking et al. (1996) undertook a population based study of people in three municipalities which surround three TV and FM radio towers in North Sydney. The health status for leukaemia and brain tumour in the three inner municipalities was compared to the health status in a ring of six outer municipalities, Figure 22.

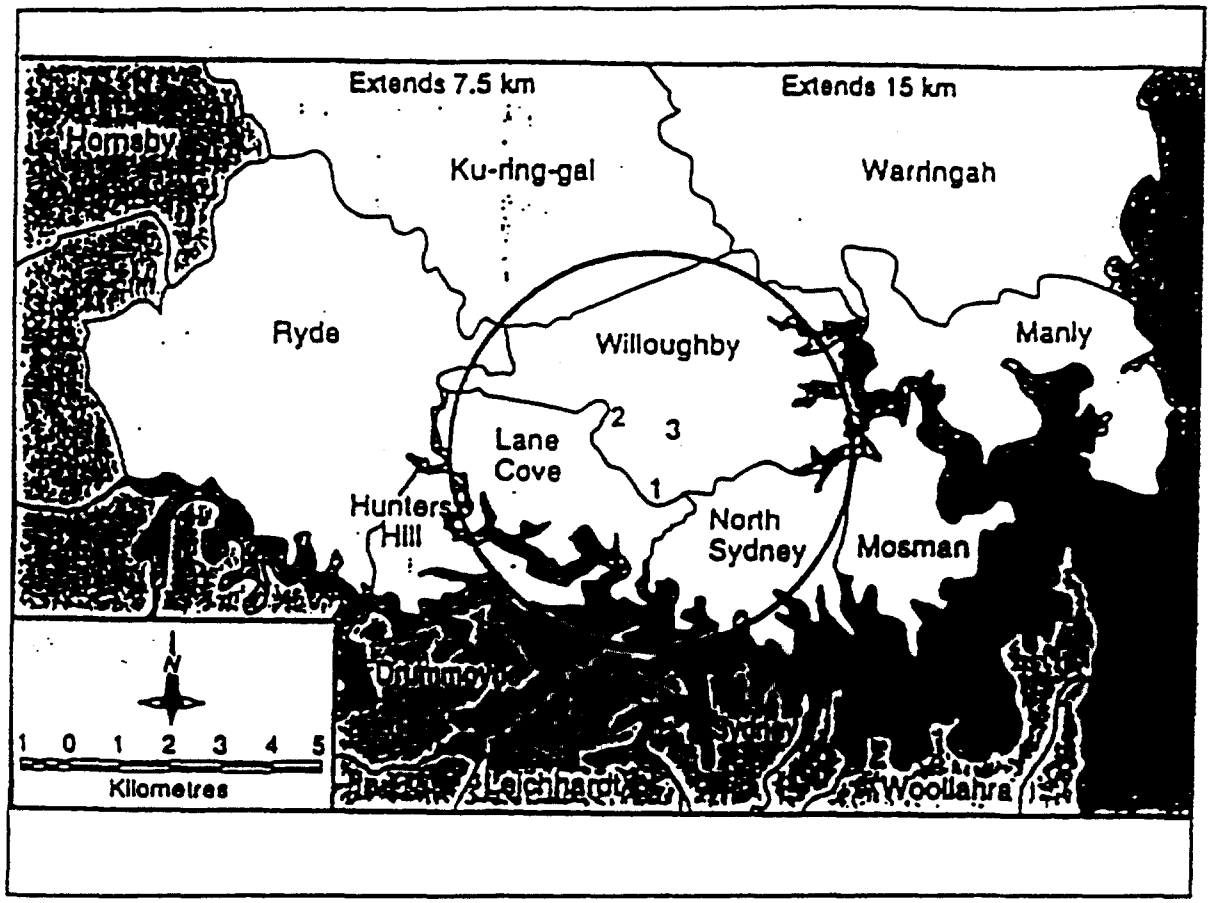

Figure 22: Municipalities in northern Sydney and the TV towers (numbered 1, 2 and 3). The circle has a 4 km radius and is for reference only. Willoughby, Lane Cove and North Sydney are the inner "exposed" municipalities, Hocking et al. (1996).

12.1.2 Effects Associated:

Among children, the rate ratio for total leukaemia incidence was 1.58 (CI: 1.07-2.34) and for total leukaemia mortality it was 2.32 (CI: 1.35-4.01). For childhood lymphatic leukaemia, the most common type, the rate ratio was 1.55 (CI: 1.00-2.41) for incidence and 2.74 (CI : 1.42-5.27) for mortality.

The exposed population compared to the New South Wales population has a non-statistically significant increase in childhood brain tumour incidence of 30% (SIR/SMR= 1.3; CI:0.7-2.3), while the "outer" group has a 20% increase (SIR/SMR= 1.2; CI:0.9-1.6). Total leukaemia incidence for all ages was 1.24 (95%CI: 1.09-1.40), Table 4.

These data clearly show the greater susceptibility of children to leukaemia in association with RF exposure than adults, Table 5.